Chapter 6

WORKERS EXPOSED TO CIGARETTE SMOKE AND STATIONARY SOURCES OF CARBON MONOXIDE

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This chapter discusses the subject of carboxyhemoglobin levels and the diseases associated with carbon monoxide exposures discussed earlier (see Chapters 2, 3 and 4). The central theme in this chapter is the influence of cigarette smoking on workers who are also exposed to stationary sources of carbon monoxide in their work environment. The problem is a complex one because the diseases that develop in these workers are usually related to their occupation. It is difficult to evaluate the sources of carboxyhemoglobin, namely carbon monoxide in the work environment, carbon monoxide from metabolism of a chemical used by the worker, and carbon monoxide from cigarette smoking. In the epidemiologic comparisons showing that the incidence of cardiovascular disease has a positive correlation with carboxyhemoglobin levels (Chapter 3), the differences in occupational exposure were not taken into account. The same criticism applies to the association between carboxyhemoglobin levels of mothers and low birth weight of their offspring (Chapter 5). This chapter discusses the carboxyhemoglobin in workers to illustrate the importance which occupational exposure could have if such exposures could be causally related to diseases of the cardiovascular and bronchopulmonary systems.

A. Exposure to Carbon Monoxide

It is important to discuss the group of workers who are positively exposed to carbon monoxide. These workers have elevated levels of carboxyhemoglobin and develop signs of acute poisoning. However, the long-term effects of carbon monoxide in workers is difficult to prove because of the concurrent exposure to other inhalants.

1. Firemen.

One major stationary source of carbon monoxide is the accidental combustion of houses and buildings. Sammons et al.[1] investigated Oklahoma City fire fighters to determine whether they had residual elevated levels of carboxyhemoglobin following occupational exposure. A control group was paired for age, weight, race, smoking habits and family history of cardiovascular and pulmonary disease. The blood samples were taken monthly for 5 months. The nonsmoking fire fighters had a mean level of 5% saturation, whereas the nonsmoking controls had a mean level of 2.3%. As a group, the fire fighters exhibited changes in enzyme activities in the blood that suggest myocardial damage.

A group of 13 fire victims showed levels of 8 to 40% with signs and symptoms of carbon monoxide poisoning: headache, weakness, confusion and restless behavior.[2] In another group of four victims, there was lactic acidemia associated with carbon monoxide poisoning.[3]

Peters et al.[4] studied the pulmonary function in 1,430 Boston fire fighters during the period of 1970 to 1972. The rate of loss in pulmonary function

observed for the entire population was more than twice the expected rate. The reductions in forced vital capacity were significantly related to frequency of fire exposures and could not be explained by differences in age, smoking habits or ethnic background. The results of this study indicate the contribution of exposure to products of combustion in the impairment of pulmonary function in fire fighters.

In a recent abstract, Kurt and Peters[5] examined the cardiac risks of the same group of fire fighters in Boston. The levels of carboxyhemoglobin were reported to be as high as 17.8% saturation. There were coincident abnormalities in serum lipids and the electrocardiogram which could not be explained by carboxyhemoglobin levels.

Firemen are exposed not only to carbon monoxide but also to products of combustion. The carbonization of plastics is a primary source of carbon monoxide. The other products of combustion have been examined acutely in animals resulting in pulmonary lesions.[6-10] There are no chronic exposure experiments to show development of heart disease. The survivors of a 1929 fire at the Cleveland Clinic were examined in 1965.[11] On the basis of the data collected, it was not possible to detect any long-term effects on the mortality experience of survivors with known exposure to carbon monoxide and other gases produced by the decomposition of nitrocellulose x-ray film at the Cleveland Clinic fire.

2. Steel Workers.

In 1929, Farmer and Crittenden[12] examined the blood of steel mill operators at the end of a working day, again, in the morning as they reported for work and finally at the end of the second working day. Their results were

as follows:

	Mean % COHb
First day 4:00 p.m.	6.26
Second day 8:00 a.m.	2.11
Second day 4:00 p.m.	7.01

The increase in carboxyhemoglobin levels in the course of a working day noted above has been confirmed by Jones and Walters,[13] Schieche et al.,[14] and Alvarez de Lugo[15] and Crosetti et al.[16] The most recent study was reported in 1974 by Butt el al.[17] who measured carboxyhemoglobin levels at the beginning and end of a single, work shift. A hundred blast furnace workers and a randomly-selected control group of 87 men with no occupational exposure to carbon monoxide were tested over a five-day period. The results permitted the comparison of smokers and nonsmokers before and after occupational exposure.

a. Beginning of shift. There were six groups differentiated according to their smoking habits, three control groups and three groups working at the blast furnace.[17] The mean and Standard deviation for each group of 21 to 45 subjects were as follows:

Categories	COHb %: mean ± S.D. Control	Blast Furnace
nonsmokers	0.9 ± 0.32	1.1 ± 0.46
smokers (<20/day)	3.3 ± 1.45	2.9 ± 2.01
smokers (>20/day)	5.0 ± 2.25	6.0 ± 2.80

The above values show a standard deviation for smokers that is five to seven times larger than that for nonsmokers, both control groups and working groups. The values for nonsmokers overlap with those of the smokers indicating the considerable range of values for smokers (see Chapter 2).

b. End of shift. For the blast furnace workers, the increase of carboxyhemoglobin levels was as follows:[17]

blast furnace	Δ of Mean before and after shift
nonsmokers	2.9
smokers (<20/day)	2.1
smokers (>20/day)	2.5

The mean uptakes of carbon monoxide for smokers are lower than that of nonsmokers. These results can be interpreted to mean that when smokers are exposed to occupational sources of carbon monoxide, their net uptake is less than that of nonsmokers (see also Chapter 1).

3. Welders.

Carbon monoxide is absorbed by welders in the course of their work. De Kretser et al.[18] measured carboxyhemoglobin levels in 60 welders and reported a range of 0 to 20%. After improving ventilation, there was a reduction to a range of 0 to 10%. Smoking cigarettes had no influence in the carboxyhemoglobin level of this group of welders. The opinions that smokers have higher levels than nonsmokers expressed by Cascini and Gazzerro[19] and by Brigatti et al.[20]

are not based on data derived from welders.

B. Diseases Associated with Occupational Exposure to Carbon Monoxide

Most workers exposed to levels above 200,000 ppm of carbon monoxide die after a few minutes of exposure.[21-23] Those who lose consciousness and recover from the poisoning may show some residual effects on the central nervous system,[24] and a few cases reports show cardiac injury with electrocardiographic abnormalities.[25-29] Most of these reports on carbon monoxide poisoning were on workers exposed directly to gas containing carbon monoxide[21-29] or to smoke.[30]

The question of long-term exposure of workers to carbon monoxide is more difficult to answer. In contradiction to the theory that a low level carbon monoxide in the work environment is dangerous to the heart, the mortality studies of gasworkers[31] and steelworkers[32] do not show a high incidence of death from heart disease (see Chapter 3).

C. Occupational Exposure Not Involving Carbon Monoxide

Although there is no disease syndrome associated with occupational exposure to low levels of carbon monoxide, there are diseases known to occur

with prolonged exposure to chemicals, dusts and particulate matter. It is important to briefly mention these occupational diseases because they are likely to appear in nonsmokers as well as in smokers. There is no data to indicate that smokers are more susceptible than nonsmokers to the following occupational diseases.

1. Chlorinated Solvents that are Metabolized to Carbon Monoxide.

Prolonged exposure to low levels of chlorinated solvents cause hepatotoxicity.[33] Dichloromethane (methylene chloride)[34, 35] and dibromomethane[36] are metabolized in the body to carbon monoxide, a phenomenon that is seen only for these two solvents. So far, the health hazard of the formation of carbon monoxide in vivo has not been fully appreciated. Smokers exposed to either chlorine gas or ethylene do not show differences in lung function compared to nonsmokers.[37, 38]

2. Hypersensitivity Reactions.

The list of substances that cause hypersensitivity or allergic response in workers has been increasing in number. The manifestations are usually in the respiratory system and are usually diagnosed incorrectly as chronic bronchitis. The substances that cause allergy of the respiratory tract of workers are as follows:

gasoline[39]

propionates[40]

insecticides[41]

enzymes for detergents[42-44]

coffee powder[45]

grain dust[46-48]

grain weevil[49]

moldy hay and moldy vegetable produce[50, 51]

fungus[52]

bird excreta and dandruff[53]

wood dust[54]

mine dust[55]

woll dust[56]

chimney dust[57]

flour and baking soda[58]

To the above list, tobacco dust can be added. Tobacco workers develop dermatitis.[59-61] Valic et al. examined the respiratory responses of 318 nonsmoking female workers in 3 cigarette factories in Yugoslavia.[62] The prevalences of chronic bronchitis and nasal catarrh were not higher among subjects exposed to tobacco dust than among the control group. A significant temporary decrease in ventilatory capacity during the work shift was recorded among the nonsmoking women. The mechanism of the reduction was not discussed. The effect is probably caused by the inhaled dust per se independent of the nicotine contained in the tobacco.

D. Practical Aspects of Occupational Exposure to Carbon Monoxide

An examination of the literature on occupational exposure of cigarette smokers indicates that, at the present time, it is difficult to identify the cause of their diseases. Some of the respiratory problems are more likely to be attributed to the work environment, such as the chemicals and dust particles which induce hypersensitivity in the workers. The carbon monoxide in the work place causes an elevation of the carboxyhemoglobin level but there is no evidence that low levels cause cardiovascular disease.

The occupational standard for carbon monoxide is under consideration by the Occupational Safety and Health Administration. The level of 50 ppm in force in the United States[63] is being questioned. A standard of 35 ppm carbon monoxide is suggested based on the reported effects of low levels of carbon monoxide on the heart and on the central nervous system, the subject covered in Chapters 3 and 4.[64] There is a definite impression that the proposed standard of 35 ppm which is calculated to keep the carboxyhemoglobin level of workers at 5% will force the dismissal of some workers who are now employed in areas containing sources of carbon monoxide. The carboxyhemoglobin levels of smokers vary substantially from individual to individual, and from day to day and from hour to hour (see Chapter 2). Those

who exceed 5% carboxyhemoglobin because of the nature of the uptake and elimination of carbon monoxide will suffer from unemployment.. Both smokers and nonsmokers who live in areas with high levels of carbon monoxide in the ambient air as well as those exposed to vehicular traffic will be excluded by the proposed standard. The new proposal has stirred a considerable amount of controversy among authorities.[65-70] Additional investigation is needed to determine the justification for changing the occupational standard to 35 ppm carbon monoxide.

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